I consider the surgical treatment of uncomplicated kidney and upper ureteral calculi safe, sane, and advisable early.

As regards calculi in the lower ureter, a proper interval should be allowed for the voluntary passage of the stone, aided by suitable ureteral instrumentation, but when it appears likely that this will not be accomplished, then at an early date one of the open operations for calculus in the lower ureter should be performed.

1401 South Hope Street.

DISCUSSION

HENRY A. R. KREUTZMANN, M. D. (2000 Van Ness Avenue, San Francisco).—Doctor Farman has given a very interesting survey of calculi in the urinary tract. A mortality of 5 per cent is very low where operations on pyonephrotic kidneys are included in this series.

Nephrotomy *per se* is a serious operation. It is greatly complicated when dealing with a pyonephrotic kidney or an associated pyelitis. Because of this infection, a secondary hemorrhage is more likely to occur. Whenever possible the calculi should be removed through a pyelotomy incision. Where the kidney pelvis is intrarenal and not hydronephrotic, there is no choice and a nephrotomy must be performed.

The question of drainage of the kidney pelvis through the nephrotomy wound with a small rubber catheter is still not settled. Some urologists advocate this method, whereas others say it increases the possibility of a secondary hemorrhage.

It is always advisable, where either ureteral or renal stone is associated with infection, to reduce the infection to a minimum by renal lavages and the use of an inlying ureteral catheter before operation.

Doctor Farman is correct in not waiting too long for a ureteral stone to pass into the bladder. It is a difficult matter to say just when to operate. If the calculus does not move, if a beginning hydro-ureter and hydronephrosis is detected, or a pyelitis sets in, one should not hesitate to operate. If, however, the stone gradually moves toward the bladder we can afford to wait for some time.

❦

WIRT B. DAKIN, M. D. (756 South Broadway, Los Angeles).—It seems to me, after careful study of these cases, that in arriving at a conclusion it is best not to take any chance; the best action taken is no action at all. I have in mind a patient about three and one-half years ago in whom there were violent bladder symptoms. A large stone and enlarged prostate were also present. There was a stone in the kidney, and after carefully checking that two or three times, waiting one-half hour for 'phthalein to come through, we found that what return we did get was more from the kidney that had the stone than from the other one that was supposed to be better. Doctor Scholl was in the hospital that morning and I asked him to see the patient. He told me to leave the kidney stone alone, and the man is still alive.

❦

WILLIAM E. STEVENS, M. D. (870 Market Street, San Francisco).—Doctor Farman is to be congratulated on the excellent results obtained in his interesting series of cases.

As in prostatic hypertrophy, preoperative and postoperative treatment is important when surgical removal of renal and ureteral calculi is required. The kidney infection should receive appropriate treatment before, and, if necessary, after operation, and ureteral strictures should be dilated.

Pyelography is of value in the determination of the location, and occasionally of the presence and size of renal calculi. Of interest in this condition is a patient seen some time ago who had a stone apparently one centimeter in diameter in the left kidney. The coating of this calculus, following the injection of sodium iodid solution, however, showed it to be two and one-half centimeters in diameter. One of the most interesting cases of nephrolithiasis that has come under my observation was that of a man with an enormous coraliform calculus which filled the entire right kidney pelvis and the calices. The left kidney contained a small calculus the size of a pea. There was no pain in the right kidney and its function was better than that of the left.

It is sometimes possible to remove a kidney stone through an incision in the upper ureter in the presence of a pelvis which is entirely intrarenal.

Some time ago I operated on a young man suffering from bilateral giant ureteral calculi. The stone removed from the left ureter weighed forty-four grams. Three stones were removed from the right ureter several weeks later. The largest of these weighed eighteen grams. The literature contains no record of a similar case.

❦

DOCTOR FARMAN (Closing).—I wish to express my thanks for the discussion of my paper.

The point raised by Doctor Kreutzmann in regard to drainage of the kidney pelvis by the introduction of a small rubber catheter through the nephrotomy incision, in my experience has proved to be a good measure. It aids in preventing postoperative pyelonephritis. Urinary fistulae, following operations upon the kidney and upper ureter, are rare.

No doubt a certain number of recurrences of stone formation can be prevented through a careful plan of follow-up treatment to reduce urine infection, decomposition, and retention in any part of the urinary tract.

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# TRAUMATIC SHOCK—ITS NEWER ASPECTS AND TREATMENT*

*By* GEORGE K. RHODES, M. D.
AND
CAROL MCKENNEY, M. D.
*San Francisco*

DISCUSSION *by Ernest H. Falconer, M.D., San Francisco; Burns Chaffee, M.D., Long Beach; Charles T. Sturgeon, M.D., Los Angeles.*

THE term "traumatic shock" is often loosely applied to any of the several collapse states which may follow an injury. These group themselves roughly into three divisions: (1) Primary shock, which develops immediately following an injury and is a condition which is nearly always caused by hemorrhage or a nervous manifestation such as syncope. (2) Very late toxic states which are usually the result of sepsis in the wounded area, secondary to virulent infection such as gangrene. (3) Secondary or true traumatic shock, occurring after a definite but not extensive time interval and not caused by nervous collapse, hemorrhage, or infection.

It is the latter state with which this paper deals.

## SECONDARY OR TRUE TRAUMATIC SHOCK

The subject of traumatic shock is still a very live one. In the course of our association with the San Francisco Emergency hospitals we have an exceptional opportunity to study this problem.

* From the Department of Surgery, University of California Medical School, San Francisco.

* Read before the General Surgery Section of the California Medical Association at its fifty-seventh annual session at Sacramento April 30 to May 2, 1928.

Traumatic shock is a general body state which is characterized by the following essential symptoms and signs:

1. There is a persistent reduced systolic and diastolic pressure associated with a rapid thready pulse and lowered pulse pressure.

2. The respirations are rapid and superficial.

3. The skin, which may be pallid, grayish, or cyanotic, is cold and moist with sweat.

4. Thirst is common, and nausea and vomiting are frequent.

5. The patient is often restless, but there is a lessened sensibility and a dulled mental state.

6. The temperature is usually subnormal.

7. Special laboratory tests reveal an increase in red blood cells and hemoglobin in the peripheral circulation. They[1] also show other evidence of decreased blood volume and volume flow.

When confronted with such a picture in a patient, it is essential to determine at once the presence or absence of acute hemorrhage as a superimposed factor. This differentiation is vital because gross hemorrhage usually demands hemostasis before the routine treatment of shock can be instituted safely.

If hemorrhage is the major factor, the clinical picture is altered in that: (1) The apathy is replaced by anxious alarm. (2) Instead of the passive immobility of shock there is restlessness and often a marked degree of muscular activity. (3) The shallow rapid respiration is replaced by deeper and more labored breathing, which is terminally aptly described as typical air hunger. (4) The examination of the peripheral blood shows a diminution in red blood cells and hemoglobin as in secondary anemia, thus differing markedly from the findings in shock. There is also a leukocytosis with a relatively high percentage of polymorphonuclear elements, and when the hemorrhage is internal there is a rapid and very marked increase in white blood cells. In patients suffering from ruptured spleen or liver, we have recorded a leukocytosis of 70,000 within one hour after injury. When the hemorrhage is secondary to intra-abdominal lesions, we usually have other associated evidences of an acute abdominal condition.

The constant derangement of physiological function in shock is one which affects the circulation and which brings about a lowered blood pressure and volume flow through loss of available blood volume from the vascular system.

### PRINCIPLES UNDERLYING PROPER TREATMENT

To treat this condition of traumatic shock in a rational manner it is essential to understand the perverted physiological mechanism underlying the production of the pathological state. It may be assumed that the lowered arterial and pulse pressure, with the resultant diminution of blood volume flow, brings about a relative tissue anoxemia. This lowered blood pressure and accompanying tissue anoxemia are accountable for the other phenomena, such as lowered metabolic rate,[6] lowered blood alkali reserve (acidosis), and loss of body heat.[2 3 4 5] If the low blood pressure with its reduced volume flow is sufficiently persistent, permanent cellular changes are brought about in the higher nerve centers,[7 8] and these ultimately result in paralysis of the vasomotor center.[9] Herein lies the great need for early treatment in shock, for once the vasomotor center has become permanently damaged by the anoxemia, vascular tone is lost. When this loss of vascular tone exists, our efforts to combat shock, particularly those endeavoring to restore fluids, will be fruitless. The problem accordingly resolves itself into an attempt to interrupt the chain of events which leads to these typical vicious circle reactions.

Satisfactory explanation of the low arterial pressure is difficult. It is here that the theoretical accounts of the initiating factor in shock widely diverge. To consider them it is necessary to review briefly the possible factors which may bring about low blood pressure. There are: (1) Weakness and failure in the pumping apparatus, the heart. (2) Increase in the size of the vascular bed, partially at least under control of the vasomotor center. (3) Loss of blood volume with associated increased viscosity of the blood.

The cardiac factor can be dismissed at once, as no evidence that the myocardium plays any part in the circulatory failure of shock has ever been adduced.[10 11 12]

The theories depending upon vasomotor paralysis with its resultant stagnation of blood in the peripheral and splanchnic areas, though long popular, are untenable in the light of recent clinical[13] and experimental evidence.[14 15] This tone is diminished or lost only after the vasomotor center has been damaged by the anoxemia to which it has been subjected. The acidosis known to be present in shock states, also has been put forward as a cause of shock. However, the acidosis can be demonstrated to follow the lowered blood pressure rather than precede it.[2 3] The anoxemia attendant upon this diminished blood flow is in all probability the cause of the acidosis rather than the result of it.[4 5 18]

The above observations leave us with the third, or blood volume reduction factor, as the sole remaining possibility. That an actual and important loss of volume in the circulating blood does in fact occur, and this probably from hyperpermeable capillaries, is forcibly shown by the work of W. B. Cannon of the World War shock commission.[1 17] In that investigation blood volume was computed by the dye and dilution methods in all stages of shock and was invariably found to be markedly below the normal figures. That this loss of volume is at the expense of the fluid portion of the blood was demonstrated by hematocrit, red blood cell, and hemoglobin readings, which showed a definite concentration of the cellular elements in the peripheral or capillary

circulation. That these changes are not so marked in blood obtained by venipuncture strongly suggests the capillary bed as the probable site of the volume loss. That the loss is of the plasma and not the water portion, is shown by the failure of the protein content in the capillary blood to increase, which it would were only water to pass from the vascular system into the tissues.[18]

The relative increase of the cellular elements augments the viscosity of the blood, which further reduced the flow.

What causes this transudation of plasma from the blood into the tissues? War data have disclosed that men with severe wounds do very much better if the injured, often crushed, member is amputated before the onset of shock. There are other observations of a like nature which indicate the wounded area as the location of the factor which initiates the systemic reaction of shock. There are but two pathways by which such an effect could travel from the focal lesion, namely, the nervous and the vascular. Some simple but illuminating work by Cannon answers this question. In a series of well-controlled experiments,[9 19 20] he induced the typical clinical and laboratory pictures of traumatic shock in animals by crushing the thigh muscles. He made a continuous graph of the blood flow and pressure before and during the experiment. The following illuminating facts bearing on the therapy for the condition were noted: The blood determinations showed a distinct diminution in volume and flow and an increase in hemoglobin and red blood cells in peripheral circulation. That these effects were not the result of nerve reaction from the traumatized leg was demonstrated by sectioning all nerve communications to the part. This procedure did not alter the results in any respect. When the vessels from the part were clamped, no shock resulted. Clamping the vessels after the induction of shock resulted in a cessation of the blood pressure fall and in recovery in many cases. Reopening the circulation to the crushed tissues resulted at once in an exacerbation of the shock signs and eventually death. Massage of the injured tissues or active motion of the fractured member abruptly aggravated the shock status. Crossed circulation experiments gave identical results in an entirely uninjured animal, without producing shock in the injured. The conclusion must be drawn that there is a toxic substance disseminated from the traumatized tissues via the blood stream which has the ability to produce that clinical picture we choose to term "traumatic shock."

Above have been briefly outlined the various contributing elements which enter into the composite entity. The nature of the toxin is not entirely clear although the evidence at hand would indicate that the state is the result of a proteose intoxication. It is known, for example, that proteoses are released from traumatized tissues.[19] It is also apparently proved that proteoses and histamin bodies will produce all the conditions seen in shock.[21 22 23] The inference that the toxin is produced by proteolytic activity is strongly suggested. Possibly substances given off by extravasated blood are likewise depressive to circulation, as blood injected just prior to coagulation does drop blood pressure. There are other clinical entities such as gas gangrene, high intestinal obstructions, extensive burns, and severe local sepsis which bring about a pathologic state identical with traumatic shock. In each of these conditions the etiologic factor is quite generally accepted as being the result of a toxic proteose absorption.

### ACCESSORY AND AGGRAVATING FACTORS

Though a toxin derived from traumatized tissues is the fundamental feature in the initiation of shock, it seldom works alone to produce this state. Usually one or more specific aggravating factors are present to augment or prolong the toxic effect. Particularly does hemorrhage before and during shock jeopardize the patient's chances for recovery. There is a point in the falling blood pressure which represents a critical level, beyond which anoxemia, acidosis, and other evidences of shock make their appearance.

In uncomplicated shock this level is a systolic arterial pressure of about 70 to 80 millimeters of mercury. When, however, the patient has suffered hemorrhage, even in amounts which alone would be totally unproductive of symptoms, the shock state is greatly aggravated so that the critical level of the blood pressure should be raised ten or more millimeters of mercury. Exposure to cold markedly endangers the patient's prospects by inaugurating a vicious circle in which the slowed circulation has already caused a drop in body heat.

The question of surgical anesthesia comes into prominence as a possible augmenting agent in shock. Ether is known to have a depressive action on the myocardium, thus introducing a cardiac element into an already embarrassed circulation. Properly proportioned and administered nitrous oxid and oxygen or local anesthesia obviates this danger. Other complicating factors are vomiting and sweating, both of which increase the fluid loss. Chest wounds with resultant impairment of respiration, jolting or motion between bony fragments which may occur in transportation, also increase the shock. Undoubtedly psychic and nervous manifestations may aggravate the approaching collapse of the circulatory system.

### PROPHYLAXIS AND TREATMENT OF SHOCK

To be effective, the treatment and prophylaxis of traumatic shock must be predicated upon the facts above noted. It must always be before us that the early application of even the simplest remedial measures is of fundamental importance. The various vicious circles which tend to establish themselves suggest certain procedures at once.

A. *Control of Hemorrhage.*—This must be done promptly and adequately, for it has been proved that individuals who have been severely

injured are often reduced to the state of shock by even a slight hemorrhage. The use of a tourniquet alone for the control of hemorrhage is to be condemned when simpler means, such as packing, pressure, or ligation, will suffice. The release of a tourniquet, long applied, is often attended by disastrous results to both the extremity and the injured individual. The tourniquet, therefore, should be reserved only for short temporary application, as during an operative procedure. (Note: The tourniquet has its ideal usage when it limits the absorption of toxic material from an extremity. In these instances when the extremity must obviously be sacrificed on account of trauma, gangrene, or sepsis, the amputation is performed proximal to the permanently placed tourniquet.)

B. *Conservation and Restoration of Body Heat.*—The association between the incidence of shock and loss of body heat has been well established. The conservation and restoration of body heat alone has frequently been efficacious in turning the tide in the patient's favor. The injured man should be surrounded immediately by dry clothing, warm blankets, and hot water bags, and during the preliminary examinations there should be the very minimum exposure of the body. When possible, hot drinks are an effective mode of contributing heat to the injured.

C. *Pain, Restlessness, and Psychic Disturbances.*—These should be controlled early by the liberal administration of opiates together with properly applied surgical dressings and psychology in the nature of reassurance.

D. *Posture.*—The elevation of the foot of the bed with so-called "shock blocks" is still a routine procedure although the rationale has been questioned.[24 25] Undoubtedly, in primary shock or syncope it is a most valuable measure.

E. *Medication.*—Symptomatic treatment is directed principally toward restoring the low blood pressure to a normal level. Vasoconstrictors, such as ephedrin, adrenalin, and pituitrin, are probably of slight value because, though they may increase the blood pressure temporarily, they fail to increase the blood volume and may, in fact, decrease it further by constricting the capillary bed. Caffein in large doses is used almost routinely as the proper stimulant for the patient in the shock state. Certain observers recommend also various cardiac stimulants of the digitalis group for these patients. The value of both caffein and digitalis is probably overestimated, as apparently there is no intrinsic myocardial fault demonstrable.

F. *Administration of Fluids.*—The effort to raise the blood pressure by forcing fluids is a more rational procedure, as these fluids act in some measure as a substitute for the lost blood plasma. The effect is transitory, however, as the added fluid quickly leaves the vascular channels through the damaged and permeable capillaries; but, though transitory, the benefit is none the less actual and often will serve to tide the patient over a critical point. When possible, the administration of fluid by mouth and rectum is simplest and most efficacious. The intravenous or subcutaneous routes, however, are always available. Lately intravenous glucose, the metabolism of which is assured by insulin, has become deservedly popular, as not only is volume thus furnished, but the cellular nourishment so greatly needed in many of these patients is provided. Particularly is this true of those patients suffering from shock in whom the pancreas temporarily seems to have ceased functioning. Blood transfusion is undoubtedly the best procedure of this nature, as the plasma so supplied, being colloidal in nature, is not so readily lost into the tissues as the more diffusible crystalloid solutions. In the opinion of most recent authors, gum acacia solutions are too dangerous to permit their use.

There is but one way to attack the fundamental cause of the shock state. The dissemination of the shock-producing toxins from the injured tissues must be prevented or limited as completely as possible. Cannon's experiments with injured extremities of animals have a practical application which should standardize the treatment of hopelessly mangled human extremities.

## SUMMARY

Accordingly, for the San Francisco Emergency Hospital service, we have endeavored to formulate a rational method of treatment for seriously injured extremities. We have combined the aforementioned routine symptomatic therapy with adequate modern surgical treatment as outlined below:

1. For the simpler comminuted fractures and for other less severe injuries, adequate splinting will suffice.

2. For the badly compounded fractures or severely lacerated extremities, thorough debridement under nitrous oxid-oxygen anesthesia, with immediate institution of Carrel-Dakin therapy, is indicated.

3. For the hopelessly mangled extremity which obviously is ultimately to be sacrificed, even more radical measures are adopted. In these cases a tourniquet is carefully and permanently applied just proximal to the traumatized tissues. The patient is then treated for traumatic shock by the various measures outlined, and the blood pressure and general status carefully watched. The patient's blood pressure must be at least 80 to 90 millimeters of mercury before we consider amputation safe or advisable. The time interval required to reach this minimum systolic pressure bears a direct relation to the adequacy of the routine shock treatment applied. It also bears a direct relation to the period which has elapsed before the application of the permanent tourniquet. The time required to prepare the patients in our series varied from two to twenty-four hours. Extended periods of time must be avoided at all hazards, for the disintegrating tissues distal to the tourniquet encourage sepsis which may rapidly surmount the tourniquet itself in cases of gas gangrene caused by *B. welchii* or similar organisms.

We are convinced that every patient who has extensive severely traumatized wounds routinely should receive immediately prophylactic inoculations of both tetanus and anaërobic sera. The polyvalent serum developed from the saccharolytic and proleolytic anaërobic cultures has proved highly satisfactory both as a prophylactic and as a therapeutic agent.

The amputation is usually of the guillotine type, rapidly performed just proximal to the tourniquet, which is left in place throughout the operation. This is most important. Indeed, this tourniquet is at no time disturbed after its original application. We have repeatedly verified clinical data, showing that even temporary release of such a tourniquet which has been long applied often results in rapid and fatal relapse into the shock state.

Results in the treatment of hopelessly mangled extremities by the outlined procedures have been most gratifying to us in a considerable number of cases. We have observed many instances of dramatic recoveries of patients who seemed almost moribund, and for whom even bilateral amputations done in accordance with the described technique were successful.

These procedures are submitted as a guide for the rational treatment of patients suffering from traumatic shock. For those with hopelessly mangled extremities, they offer a very practical mode of treatment.

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DISCUSSION

ERNEST H. FALCONER, M. D. (384 Post Street, San Francisco).—This is an excellent résumé of the present status of shock because it deals with the physiology of the condition. Once the underlying factors in the production of shock are understood it becomes readily amenable to treatment if the simple measures mentioned by Doctors Rhodes and McKenney are promptly carried out. It is worth while emphasizing again the fact that if the vasomotor center in the brain is deprived for more than one hour of an adequate blood supply and, of course, oxygen supply, which is the important thing, it becomes nearly a hopeless task to bring an individual out of the shock state. The reason being that the vasomotor center suffers permanent damage rapidly when its blood supply is cut off by the extremely low blood pressure and poor circulation of the shock state. It is extremely important, therefore, to watch the blood pressure. When critical blood pressures are present never operate on a patient under ether anesthesia. The simpler resuscitation measures as external heat, fluids intravenously, hot drinks, morphin in large doses (one-half grain), will often serve to restore the pressure. If it continues to drop below the critical point, prompt recourse to blood transfusion is indicated. The discusser used gum-salt solution extensively in shock and resuscitation work during the World War and never noted any untoward results, but apparently this has not been the general experience.

BURNS CHAFFEE, M. D. (917-21 Security Building, Long Beach).—The essayists have presented a very practical paper on an ever-important subject—automobile accidents alone furnish too many cases of traumatic shock. As has been stated, shock may be caused experimentally by one of several conditions; however, in man shock is generally the result of several factors. I do not believe that you can exclude nervous collapse or absorption of toxic substances, nor entirely rule out hemorrhage in every case. If hemorrhage is the primary factor, blood transfusions bring about a rapid recovery. Not infrequently the leukocyte count may be twenty thousand or even higher and in some cases of moderate shock in which no hemorrhage exists, and in the absence of crushed tissue; the increased count is probably due to absorptions of toxins resulting from more or less trauma to the entire body. Evidence at hand proves beyond a doubt that surgical shock is essentially due to intoxication by materials derived from damaged tissues and no doubt there is a difference in individuals' ability to tolerate the toxic substances whether they be proteoses or histamine bodies. This may explain why certain individuals are more susceptible to traumatic shock than others.

CHARLES T. STURGEON, M. D. (1930 Wilshire Boulevard, Los Angeles).—Whatever the absolute cause of shock may be, the end result is deoxygenation of the body tissues which results from diminution of blood volume in circulation. The loss is due to stagnation of the blood in the capillaries. Hemorrhage and low blood pressure are frequent factors. The rationale of the treatment, therefore, is to restore the volume in circulation and to raise the blood pressure.

Blood transfusions are superior to any other form of treatment in restoring the blood volume whether hemorrhage is or is not a factor in the production of the shock. Next in value is a glucose solution—10 per cent glucose in 1000 cubic centimeters of salt solution plus insulin. Next in value is salt solution, and although salt solution is the least valuable of the three mentioned, still it is always procurable and easily prepared. If for some reason it is impossible to give a blood transfusion or glucose at once, the salt solution should be given.

The method of administration should be the intravenous route. If the solutions are well prepared and given slowly, there is no danger of any heart com-

plications or severe reactions. The subcutaneous and rectal method are too slow and indefinite when dealing with a very sick patient.

To raise the blood pressure the best thing we have found has been the use of ephedrin sulphate. In some cases where the pressure has been extremely low we have not hesitated to use it intravenously, and repeated it as often as necessary because it is definitely known that patients with a low blood pressure of 80 or less which has existed for more than four or five hours rarely recover.

If when surgery is necessary, such as an amputation, etc., the question arises what type of anesthetic is best to use. Spinal anesthetic does wonderfully well for this type of patient although it has a tendency to depress the blood pressure slightly. If ephedrin sulphate has been used just before the anesthetic and given during the operation if the blood pressure drops perceptibly, the operation can be performed without any risk to the patient. I think spinal is an ideal anesthetic.

The more we understand the pathology of shock the clearer our line of treatment will be.

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## THE PSYCHONEUROTIC PATIENT AND THE CLINICIAN*

*By* WILLIAM H. BARROW, M. D.
*San Diego*

DISCUSSION *by Arthur L. Bloomfield, M. D., San Francisco; V. R. Mason, M. D., Los Angeles; C. M. Haviland, M. D., San Diego.*

PATIENTS are of two types, one group presenting symptoms which are clear-cut and apt to be minimized; the other group with complaints which seem to spread out like the waves on a pool of water into which a rock has been thrown and where though the primary disturbance is really slight the turmoil of the waters is great.

It is inevitable that, under the stress and strain of our modern social and industrial existence, the psychoneuroses and neurasthenias should loom large as clinical entities and as outstanding factors in other diseased conditions. Review of medical literature indicates that there has of late been a reawakening of interest which comes as a reaction against a practice of medicine that here and there was becoming too cold-bloodedly scientific; a system where in the maze of scientific data it was possible to forget that the patient was a sentient human being who had a soul as well as a body, and that the reactions of the one were as important as the reactions of the other. Carrying on the discussion where it was left off in the literature by Weir Mitchell and Osler many years ago, there is of more recent date an outstanding monograph by the late Francis Peabody.[1] He pointed out that in order to treat a case of any sort intelligently and adequately the doctor must know the patient through and through; that disease in man is never the same as disease in an experimental animal; that its manifestations are different even in different individuals, and that disease "at once affects and is affected by . . . emotional life." In a symposium at a recent meeting at the American Medical Association, Woodyatt, Hunt, Foster McLester, Neilson, White, Einhorn, Kilgore, and Rosenow[2] discussed the interrelation of somatic and psychic disturbances and emphasized the fact that disease, be it functional or organic, must be studied and treated with both physical and psychic factors in mind. There is hardly a medical convention today without one or more papers on the functional or so called affective disorders.

In all of this discussion emphasis has been laid on etiology and diagnosis rather than on treatment. The purpose of this paper is to approach the problem from the point of view of the management of these cases insofar as such management falls within the realm of the clinician and general practitioner.

### THE PSYCHIATRIST AND THE GENERAL PHYSICIAN

In this day of specialization it might at first seem most expedient to refer these cases to the psychiatrist, but such a disposition is not generally satisfactory. The neurasthenic patient seldom consults his doctor for nervousness or mental symptoms, but rather for indigestion or headache or palpitation of the heart or for any one of a number of other physical ailments, and he resents being told that his trouble is all "nerves" and objects to being sent to a man who treats only "nerves." A patient who had had severe headaches all her life recently told me that she had been referred to a psychiatrist because there was nothing organically wrong with her and that this psychiatrist had psycho-analyzed her and told her that she was a Peter Pan type of individual, that she would have been all right if she could have remained a child, and that it was a mistake for her to have assumed the strain and obligations of married life and motherhood. "Well," she said in a tired way, "That may all be true, but what am I going to do about it now, and—I still have my headache." The patient comes to us not only for a diagnosis but for treatment of his symptoms, and where these symptoms are related to functional or organic physical disorders more can usually be accomplished by the patient's own physician than by the psychiatrist alone. Psychoneurosis is, of course, to be differentiated from true psychosis, pure hysteria, anxiety neurosis, and the other purely psychic disturbances which fall distinctly within the realm of the psychiatrist.

### SCOPE OF THIS DISCUSSION

This discussion is limited to that large class of cases included of late under the broad term of psychoneurosis, in the past more commonly called neurasthenia; a condition where the chief complaint is usually physical, where the subjective symptoms are usually out of all proportion to the physical findings, and where there is always an underlying and usually obscure mental or psychic disorder, based on some environmental or circumstantial conflict.

It would seem that in these cases the somatic manifestations are due to organic or functional disorders, the symptoms of which are exaggerated by the lowering of the patient's threshold

* Read before the eighteenth semiannual meeting of the Southern California Medical Association, April 5, 1929.